

Appl. No. 10/035,344  
Amdt. dated Jan. 28, 2005  
Reply to Office Action of Dec. 6, 2004

### REMARKS

Claims 1, 46, and 48-50 are pending in the application. Claims 2-45 and 51-116 were canceled as a result of the restriction requirement. Claims 1 and 46 through 50 are rejected. Applicants have amended claim 1 and respectfully request reconsideration of the application as amended herein.

### In the Claims

Claim was amended to clarify that the proteins complexes of the invention include those having proteins 90% or more identical to the respective "wild-type" protein or fragment. Support for this amendment is found in the as-filed specification at e.g., paragraph [0056]. No new matter is added as a result of this amendment.

### **35 U.S.C. § 101 Claim Rejections**

Claims 1 and 46-50 are rejected under 35 U.S.C. § 101 because the claimed invention is allegedly not supported by a specific, substantial and credible asserted utility or a well established utility.

As set forth on pages 2-3 of the Office Action, it is alleged that Applicants have not disclosed an actual and specific significance which can be attributed to the protein complex. Applicants respectfully disagree and direct the Examiner's attention to paragraph [0019] of the specification which discloses that AKT1 and AKT2 are involved in control cell proliferation and apoptosis. This disclosure is supported by numerous references cited in paragraph [0019] and supported by the fact the AKT pathway is a major therapeutic target for cancer. Because of the role of AKT in regulating apoptosis and AKTs involvement in cancer and neurodegeneration, Applicants asserted in the as-filed specification that protein-protein interactions with AKT are therapeutic targets. This assertion is further supported by a recent literature reference demonstrating the same interaction between protein kinase B (AKT1) and periplakin (PPL). Van den Heuvel *et al.* (J. Cell Science 115:3957 (2002) cited herewith in an IDS) report that the interaction of periplakin with AKT1 is important in regulating AKT1 activity. As mentioned in the specification and as was well known to the skilled artisan at the time of filing of the

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application, dysregulated AKT activity is a hallmark of several disease states including cancer and neurodegenerative disorders. For example, Ozes et al. (of record) describes the role of AKT1 signaling in suppressing apoptosis. Additionally, Mitsuuchi et al. (of record) teaches AKT1 and AKT2 activity are both implicated in response to marketed cancer therapeutics (cisplatin and paclitaxel). Thus, the literature clearly implicates AKT1 and AKT2 activity to pathways related to regulating apoptosis which is specifically related to cancer, neurodegeneration, and other diseases.

It is even alleged in the instant office action that "There is little doubt, after complete characterization, this protein [complex] will probably be found to have patentable utility." The Patent Office did not issue an § 101 rejection in the first office action and now is admitting that there is little doubt that the complex will be found to have patentable utility. According to the Patent Office's analysis, it is more likely than not that the disclosed protein complexes have utility under 35 U.S.C. § 101.

Substantial evidence of record indicates that the disclosed protein complexes have utility. First, the baits chosen to discover the novel interactors disclosed herein were chosen for their known involvement in a disease related pathway, in this case apoptosis, neurodegeneration and cancer. Second, others have since confirmed the utility of protein-protein interactions with AKT and specifically the AKT1:Periplakin interaction. Third, the PTO admits on record that there is little doubt that these protein complexes will be found to have patentable utility – indicating that it is more likely than not that complexes have utility. In view of the evidence of record indicating a substantial specific and credible utility for the protein complexes under examination, Applicants respectfully request withdrawal of this rejection.

### **35 U.S.C. § 112 Claim Rejections**

Claims 1 and 46-50 are rejected under 35 U.S.C. § 112, first paragraph, as allegedly failing to adequately teach how to use the instant invention. Since Applicants have disclosed a specific, substantial and credible utility, as discussed above, this rejection is rendered moot. Applicants therefore respectfully request withdrawal of this rejection.

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Claims 46 through 50 stand rejected under 35 U.S.C. § 112, second paragraph, as allegedly being indefinite for failing to particularly point out and distinctly claim the subject matter which Applicants regard as the invention. As set forth on page 3 of the Office Action, it is alleged that since claim 1 recites isolated protein complexes and claim 47 recites *in vitro* methods, it is not clear what other methods can be used save *in vitro*. In order to expedite prosecution of this application and without acquiescing to the propriety of the rejection, Applicants have canceled claim 47. In view of the claim cancellation, Applicants submit the rejection is rendered moot and respectfully requests its withdrawal.

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### CONCLUSION

Claims 1, 46, and 48-50 are believed to be in condition for allowance, and an early notice thereof is respectfully solicited. Should the Examiner determine that additional issues remain which might be resolved by a telephone conference, he is respectfully invited to contact Applicants' undersigned attorney.

Respectfully submitted,



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Date: January 28, 2005

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